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Latent Difference Score Approach to Longitudinal Trauma Research

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In this article, the authors introduce a latent difference score (LDS) approach to analyzing longitudinal data in trauma research. The LDS approach accounts for internal sources of change in an outcome variable, including the influence of prior status on subsequent levels of that variable and the tendency for individuals to experience natural change (e.g., a natural decrease in posttraumatic stress disorder [PTSD] symptoms over time). Under traditional model assumptions, the LDSs are maximally reliable and therefore less likely to introduce biases into model testing. The authors illustrate the method using a sample of children who experienced significant burns or other injuries to examine potential influences (i.e., age of child–adolescent at time of trauma and ongoing family strains) on PTSD symptom severity over time.

Researchers interested in posttraumatic stress disorder (PTSD) and other mental health consequences of exposure to traumatic events commonly impose a longitudinal perspective to their work. Variables are frequently organized according to a time-based classification, some variation on the categories of pretrauma, peritrauma, and posttrauma, with the goal of elucidating risk for, resilience against, and/or recovery from the deleterious effects of trauma exposure. Even when the data at hand are cross-sectional and retrospective (e.g., Fontana & Rosenheck, 1993; Foy, Resnick, Sipprelle, & Carroll, 1987; Green, Grace, Lindy, Gleser, & Leonard, 1990; King, King, Foy, Keane, & Fairbank, 1999), we "do the best we can" to reconstruct a process that reflects a longitudinal course indexed by a

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highly distressing life event or set of circumstances. In the cross-sectional design with retrospective reports of prior status, of course, there is always the threat of ambiguity about the direction of the chain of causality (King & King, 1991), owing to a myriad of concerns about memory and faulty recall, attempts to assign meaning to one's current mental state, and other subjective biases that may influence perceptions and appraisals of past events (e.g., Brewin, Andrews, & Gotlib, 1993; Dohrenwend, Dohrenwend, Dodson, & Shrout, 1984; McNally, 2003).

To counter such concerns, more and more trauma researchers (e.g., Koss & Figueredo, 2004; Saxe, Stoddard, Hall, et al., 2005; Shalev et al., 1998) have used longitudinal designs in which the issue of temporal precedence and the direction of causality may be better addressed. In most cases, trauma victims have been recruited at the time of or shortly after exposure and repeatedly assessed at targeted intervals for some period thereafter, albeit with accounts of pretrauma status still mainly retrospective. The logical advantage is the separation in time of score on a predictor (e.g., trauma exposure measured at or immediately after the event) and later score on a dependent variable or outcome of interest (e.g., PTSD), thereby alleviating some concern about one's condition influencing reports of earlier experiences. However, before drawing inferences about the effect of such an external predictor (trauma exposure) on the outcome of interest (PTSD), one might need to consider what can be characterized as internal mechanisms that influence standing on the outcome variable.

Using the argument of Gollob and Reichhardt (1987), a simple model in which an outcome variable, PTSD, is regressed on a putative cause, trauma exposure, fails to take into account one's preexisting status on PTSD at the time of the traumatic event. Indeed, in the trauma literature, revictimization is not uncommon (e.g., Bremner, Southwick, & Charney, 1995; King et al., 1999); many individuals experience repeated traumatic events, and thus the effect of any single event should be evaluated in light of possible existing symptomatology. Therefore, the direction of causality may not be fully unambiguous. Gollob and Reichhardt strongly recommended the use of a statistical control of one's status on the dependent variable at the initial assessment when evaluating the effect of the predictor on this variable at the later assessment. By including prior status on the dependent variable in the regression model, one is controlling for a first internal mechanism, autoregressive effects; the nature of the resulting partialled relationship becomes one between the predictor (trauma exposure) and change in status on the outcome (PTSD). Change in status, in this case, is defined as the difference between one's actual measured status on PTSD on the subsequent occasion and that predicted from one's status on PTSD at the initial occasion. In effect, from the perspective of the predictor (trauma exposure at the initial assessment), the outcome is residual PTSD (the difference between observed and predicted PTSD at the later assessment). This strategy reflects the logic of the cross-lagged panel design, where two or more variables are assessed on two or more occasions to determine the direction of causal influence (see trauma studies by Erickson, Wolfe, King, King, & Sharkansky, 2001; King et al., 2000; King, Taft, King, Hammond, & Stone, in press; Schell, Marshall, & Jaycox, 2004). It should be noted, however, that the use of residual scores as indicators of individual differences has been criticized (e.g., Rogosa, Brandt, & Zimowski, 1982; Rogosa & Willett, 1985; Willett, 1989).

Although partialling the effect of prior status on the dependent variable may clarify the interpretation of the relationship between a predictor and a subsequent score on this outcome, ambiguity in the interpretation of the predictor–outcome relationship may still be present. The reason is that analysis of time-series or panel data, that is, repeated assessments of a variable over time, assumes what is typically called *stationarity*. Stationarity simply indicates that the variance of the variable that is repeatedly assessed is equal across time and that any changes related to the mean are irrelevant. To the extent that this assumption of stationarity is not met, there may be confusion in the interpretation of the predictor–outcome association. In the realm of trauma research, longitudinal tracking of trauma victims has revealed a general trend toward wellness (Gilboa-Shectman & Foa, 2001; King, King, Salgado, & Shalev, 2003; Koss & Figueredo, 2004) and hence, a natural course, on average, for a decline in PTSD and other

symptomatology postexposure. Thus, the internal mechanism of nonstationarity or natural change also needs to be considered and addressed in any longitudinal analysis of time-series trauma data. To our knowledge, no longitudinal trauma research employing the cross-lagged panel methodology has attended to nonstationarity as a potential confound.

The purpose of the present study is to introduce the latent difference score (LDS) model for dynamic change (Hamagami & McArdle, 2001; McArdle, 2001; McArdle & Hamagami, 2001) and demonstrate some uses in longitudinal trauma research. In the section below, we begin with a discussion of difference scores, the key elements in the LDS model. We follow with a description of the model's major components and how they accommodate autoregression and nonstationarity. We then demonstrate the use of the LDS approach with longitudinal data from a study of children who experienced burns or other injuries (Hall et al., 2006; Saxe et al., 2001; Saxe, Stoddard, Chawla et al., 2005; Saxe, Stoddard, Hall et al., 2005; Stoddard et al., 2006). Following commentary on the utility of the approach in trauma research, we summarize and briefly present the assumptions, limitations, and data requirements of the method.

DIFFERENCE SCORES

Difference scores, also referred to as *gain scores* or *change scores*, are typically calculated by subtracting the score on a variable at one assessment occasion from the score on this variable at a later and usually adjacent assessment occasion. A single difference score would be designated as simply

$$D_i = Y_{1i} - Y_{0i}, \tag{1}$$

where D_i stands for a difference score for person i, Y_{0i} is person i's observed score at the initial assessment, and Y_{1i} is person i's observed score at the subsequent assessment.

For many years, the research community has been warned of the dangers of relying on difference scores to document change, with perhaps the most heeded call for abandoning difference scores coming from Cronbach and Furby (1970). In their influential article, these researchers presumed the true score did not change and then asserted difference scores to be highly unreliable. Since reliability is a prerequisite for validity (Ebel, 1961), the previous assumptions imply that difference scores have zero validity. The basis for asserting unreliability may be found in the formula

$$r_{DD} = \frac{r_{00} + r_{11} - 2r_{01}}{2(1 - r_{01})}, \tag{2}$$

where r_{DD} is the reliability of the difference score, r_{00} is the reliability of the measure at the initial assessment, r_{11} is the reliability of the measure at the subsequent assessment, and r_{01} is the correlation between scores on the two assessments. According to this formula, as the correlation between scores on the two assessments (r_{01}) increases, the reliability of the difference (r_{DD}) decreases, other factors held constant. But, as noted by Nesselroade & Cable (1974), and repeatedly by others (e.g., Williams & Zimmerman, 1996), Equation 2 fails to consider the potential for growth indicated in the dispersion or standard deviations of the scores on the two assessment occasions. Thus, a more complete representation of the formula is

$$r_{DD} = \frac{\frac{SD_0}{SD_1} r_{00} + \frac{SD_1}{SD_0} r_{11} - 2r_{01}}{\frac{SD_1}{SD_0} + \frac{SD_0}{SD_1} - 2r_{01}} \tag{3}$$

Here, the symbols represent the same elements as in Equation 2, except there are additional elements, the ratio of the standard deviation of scores at the initial assessment (SD_0) to the standard deviation of scores at the later assessment (SD_1), and its inverse, the ratio of the standard deviation of scores at the later assessment (SD_1) to the standard deviation of scores at the initial assessment (SD_0).

Opponents of difference scores have argued that these ratios of standard deviations should always approach 1.00, because classical test theory, where growth is not considered, assumes equal standard deviations over assessments using the same measure. (Thus, SD_0/SD_1 and SD_1/SD_0 are not present in Equation 2.) However, according to Equation 3 where growth is allowed, the more the value of these ratios deviate from 1.00, the greater the possibility of acceptable difference score reliability (r_{DD}). Again, as noted by Cronbach and Furby (1970), the smaller the

correlation between scores on the two occasions (r_{01}), the greater the chance for acceptable difference score reliability (r_{DD}). That is, mathematically, as the value of r_{01} in Equation 3 decreases, there is obviously an increase in both the numerator and denominator (note the subtraction involving the $2r_{01}$ term), but the effect is an increase in the value of the overall r_{DD} ratio. Researchers supporting the use of difference scores (e.g., Collins, 1996; Nesselroade & Cable, 1974; Nesselroade & Baltes, 1979; Rogosa & Willett, 1983, 1985; Sharma & Gupta, 1986; Williams & Zimmerman, 1996) have pointed out that actual study data typically suggest discrepancies in the values of standard deviations, due to, for example, fan spread representing differential developmental change or ceiling effect resulting from a successful intervention (see references in McArdle & Epstein, 1987; McArdle & Woodcock, 1997). In essence, the emphasis should be on individual differences in change scores and not individual differences in scores themselves (Rogosa & Willett, 1983; see Collins, 1996, for a clear graphical presentation of this issue). Therefore, reliability of difference scores is a more complex matter than previously believed, and worries about universal unreliable change may be overrated and unfounded.

LATENT DIFFERENCE SCORE MODEL

Let us develop a structural equation model for LDSs. Reference to Equation 3 reveals that optimizing the reliabilities of components of the difference score (r_{00} and r_{11}), other things held constant, will optimize the reliability of the difference score (r_{DD}). Accordingly, the LDS model begins with the partitioning of observed scores:

$$Y_0 = y_0 + e_0.$$

and

$$Y_1 = y_1 + e_1, \tag{4}$$

where Y_0 and Y_1 are observed scores for an individual[1] at the initial and subsequent assessments, respectively, y_0 and y_1 are true scores, and e_0 and e_1 represent measurement error. Being true scores, y_0 and y_1 are perfectly reliable. We can define the difference between these two latent variables for a person by first asserting that y_1 is equal to y_0 plus some change in y_0. If we symbolize this change as LDS_{y1}, then

$$LDS_{y1} = y_1 - y_0. \tag{5}$$

LDS_{y1} is the latent difference score, composed only of the two reliable parts.

It is not possible to estimate independently the true score and error variance when there are only two time points. To coincide with the example to follow, assume a model with four repeated assessments on a dependent variable of interest (e.g., observed scores Y_0, Y_1, Y_2, and Y_3 on a continuous measure of PTSD symptom severity); in effect, the model can be extended to any number of repeated assessments. As before, the observed scores are partitioned into their true (y_0, y_1, y_2, and y_3) and error (e_0, e_1, e_2, and e_3) elements, and three sequential LDSs may be defined in terms of their associated concomitant and antecedent latent variables:

$$LDS_{y1} = y_1 - y_0,$$
$$LDS_{y2} = y_2 - y_1,$$

and

$$LDS_{y3} = y_3 - y_2, \tag{6}$$

Figure 1 portrays such a model using a path graphic representation (McArdle & Boker, 1990; McArdle & Hamagami, 2001). Importantly, this model has features that take into consideration the aforementioned internal sources of change, autoregressive effects, and nonstationarity. To recapitulate, autoregressive effects concern the effect of one's score from a prior assessment of a variable on subsequent scores on that variable. To the extent that later scores are affected, change in scores would likewise be affected. Paths labeled *a* in Figure 1 depict the autoregressive effect on the LDS: from y_0 to LDS_{y1}, from y_1 to LDS_{y2} and from y_2 to LDS_{y3}. The *a*s are simply coefficients for the regression of LDS_{y1}, LDS_{y2}, and $LDSy_3$ on y_0 y_1, and

[1] Here and for the remainder of the article, we have dropped the *i* subscript indicating an individual's score to simplify presentation.

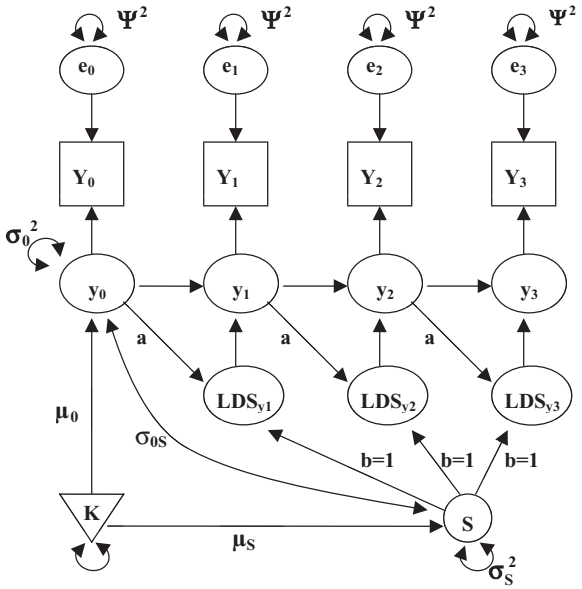

Figure 1. Ψ^2 = unique variance associated with measurement error; $e_0 - e_3$ = error scores across the four assessment occasions; $Y_0 - Y_3$ = observed scores across the four assessment occasions; σ_0^2 = variance of latent initial status; $y_0 - y_3$ = true scores across the four assessment occasions; a = proportional change coefficient; $LDS_{y1} - LDS_{y3}$ = latent difference scores; b = constant change coefficient; μ_0 = mean of latent initial status; σ_{0S} = covariance between slope factor and latent initial status; K = constant; μ_s = mean of latent slope factor; S = latent slope factor; σ_s^2 = variance of latent slope factor; *Boxes* = observed or manifest variables; *Circles* = unobserved or latent variables, including measurement errors or residuals; *Single-headed arrows* = directional associations, as in regression coefficients; *Double-headed arrows* = variances or covariances; *Triangle* =depiction of means and intercepts. Unlabeled single-headed arrows assume a regression weight of 1, and labeled arrows correspond to parameters potentially to be estimated.

y_2, respectively. The model accommodates nonstationarity or natural change by postulating a slope latent variable (s) and designating its effect on the LDSs by paths labeled b: from s to LDS_{y1}, from s to LDS_{y2}, and from s to LDS_{y3}. The as are called *proportional change coefficients* and are typically, though not necessarily, constrained to be equivalent over the series. The bs are called *constant change coefficients* and are typically, though not necessarily, all constrained to a value of 1 (as they are in the figure). In the structural equation model for LDSs, measurement error usually is constrained to be equivalent over all of the assessments.

With both proportional and constant change influences, the model is labeled as a *dual change* LDS model, with change a function of both autoregressive effects and natural growth. Thus, for the dual change model in Figure 1, where the proportional change coefficients (as) are equal and the constant change coefficients (bs) all equal 1, we can define the key elements, the LDSs, as follows:

$$\begin{aligned} LDS_{y1} &= ay_0 + 1.00s \\ LDS_{y2} &= ay_1 + 1.00s \\ LDS_{y3} &= ay_2 + 1.00s \end{aligned} \tag{7}$$

The dual change LDS model depicted in Figure 1 has seven parameters to be estimated: two means, the mean of the first latent variable score or initial status (μ_0) and the mean of the constant change variable (μ_s); the variances of each of these (σ_0^2 and σ_s^2); the covariance between the two (σ_{0s}); a single value for the proportional change coefficient (a); and a single value for the estimate of measurement error (Ψ^2).

Though both proportional change (a), and constant change (b), are in Figure 1, one or the other or both may not be necessary to explain change over time. If nonstationarity is not a relevant feature of the data, then the constant change effect is not necessary for good fit of the model, and the model reduces to a proportional change model. That is, the slope component drops out of Equation 7, and internal change would be solely a function of prior status. If a full autoregressive model is desired, then residual terms on the latent difference scores are needed as well (McArdle, 2001). In like manner, if there is no autoregressive effect, the model could revert to just a constant change model. The a component would drop out of Equation 7, leaving internal change as only a function of natural growth. It is also possible that neither of these internal mechanisms influence change, with both proportional and constant change effects absent.

In summary, we have proposed a LDS model to portray the longitudinal course of a variable. The general trend

over time has been decomposed into a series of change segments, like taking a curvilinear growth curve and disaggregating it into a series of connected linear "slices." As will be demonstrated in the next section, each of these optimally reliable segments (representing change from one specific time point to the next identified time point) has the potential of being an outcome variable to be explained or predicted. In turn, by incorporating the internal mechanisms of proportional change and constant change, we can more readily arrive at an unambiguous interpretation of the influence of hypothesized external variables. Such external variables may be fixed markers (Kraemer et al., 1997), constant or enduring time-invariant characteristics of the person or environment (e.g., gender, genotype, intensity of exposure to the cardinal event, age at time of exposure), or they may be time-varying risk factors, able to take on different values over time (e.g., social support, financial resources, cardiovascular reactivity; post-trauma family strains; King, Vogt, & King, 2004; Vogt, King, & King, in press).

LATENT DIFFERENCE SCORE DEMONSTRATION USING PTSD, AGE, AND FAMILY STRAINS

The data for this demonstration come from a program of research conducted by Saxe and colleagues (Hall et al., 2006; Saxe et al., 2001; Saxe, Stoddard, Chawla et al., 2005; Saxe, Stoddard, Hall et al., 2005; Stoddard et al., 2006). The sample is comprised of 190 male (70%) and female (30%) children and adolescents, aged 4 to 18, who were admitted to the emergency room of two Boston-area hospitals with burns or other serious injuries. Data also were obtained from parents or guardians. Assessments were targeted to take place upon hospital admission, 3 months after the event, and 1 year after the event. For a subsample of 40 children and adolescents, an assessment at 6 months, and for a subsample of 8 children and adolescents, an assessment at 18 months, were scheduled. Posttraumatic stress disorder and depression were primary outcome variables. In addition to an array of demographic characteristics, the research included measures of the severity of the trauma, prior trauma, child behavior problems, family strains, additional negative life events following the injury, and various biobehavioral markers.

We illustrate the LDS approach by addressing the following question: In what ways do age of the child or adolescent at the time of trauma and fluctuations in family strains following the traumatic event influence changes in PTSD symptom severity over time? Saxe's measure of PTSD symptom severity was the Child PTSD Reaction Index (Pynoos & Eth, 1986), which was administered to the participating children and adolescents across all assessment occasions. Using this 20-item semistructured interview, respondents were asked to rate the frequency of their symptoms on a 5-point Likert-type scale (1 = *never* to 5 = *most of the time*), and total scores were computed for analysis. The second measure for this demonstration was the Family Strains Scale, part of a larger instrument, the Family Inventory of Life Events and Changes (McCubbin & Patterson, 1996) intended to assess the degree of family stress. The Family Strains Scale consists of 10 items asking about family conflict, disruptions, and pressures, and was administered to parents and guardians at each assessment occasion. On the first assessment occasion, it referenced events and circumstances over the past year; at subsequent assessment occasions, the reference was to the period since the last assessment. Each item was accompanied by a yes/no response format, with yes responses differentially weighted and then divided by 10 to yield a composite score. Age of the child or adolescent at the time of the trauma was indexed in years. Thus, age was a time-invariant covariate of PTSD, and family strains was a time-varying covariate of PTSD.

There are many ways to investigate an appropriate or optimal group basis for timing in structural equation modeling. Applying the time structuring procedures detailed by King et al. (2006) as a precursor to the analyses, four time classes were created (0–80 days, 81–160 days, 161–240 days, and >240 days), and scores on the repeated assessments of PTSD and family strains then were sorted into their proper time classes according to the actual number of days from the traumatic event to the specific times of assessment. Thus, for example, if data were collected for

a participating child or adolescent and parent or guardian on Day 3 after the traumatic event, then again on Day 92, and then again on Day 250, PTSD and family strains' scores would be placed in the first, second, and fourth time classes, respectively, and the third time class would be assigned a missing code for both variables. Indices of central tendency and variability for the distributions of the number of days since the trauma were (a) Time Class 1, $M = 5.37$, $Md = 2.00$, $SD = 9.52$, $n = 154$; (b) Time Class 2, $M = 110.07$, $Md = 106.00$, $SD = 17.10$, $n = 85$; (c) Time Class 3, $M = 199.57$, $Md = 195.50$, $SD = 18.75$, $n = 30$; and (d) Time Class 4, $M = 390.66$, $Md = 375.50$, $SD = 45.82$, $n = 59$. For further information on methods for preparing time-based data for structural equation modeling, see King et al. (2006).

Before addressing the main question of how age and family strains influence changes in PTSD, we first determined which LDS model (the full dual change, proportional change, or constant change) best characterized dynamic process separately for each of the two time-varying variables, the time-varying outcome, PTSD, and the time-varying covariate or predictor, family strains. In this regard, for each, we began with the dual change model and inspected fit indices and parameter estimates for this model, to ascertain whether it was sufficient or whether one of the other alternative models might be more appropriate. We then evaluated the influence of the time-invariant age variable on changes in PTSD, followed by a model in which all three variables (PTSD, age, and family strains) were included. Data analyses were conducted with the Mplus software program (Muthén & Muthén, 2006).[2] The full information maximum likelihood estimator was selected for incomplete data under the missing at random assumption (McArdle, 1994; Schafer, 1997).

Table 1 displays the means, standard deviations, and intercorrelations estimated for PTSD and family strains assessed over the four occasions, plus the age variable. Consistent with expectations, average PTSD symptom severity scores tended to decline over time. Average family strains scores, on the other hand, decreased from Time Class 1 to Time Class 2, but increased from Time Class 2 to Time Class 3, before dropping again in Time Class 4. The correlations among the assessments of PTSD seem to follow a pattern in which associations between time-adjacent assessments were higher than associations between those that were more distal. In contrast, for family strains, the pattern appeared opposite, with some of the stronger associations between assessments that were more distal. Age was minimally correlated with family strains, but displayed some relationship to the assessments of PTSD, albeit not consistent in sign or value.

Table 2 shows results of initial models involving PTSD. The goal was to determine the best fitting model for change in PTSD, and we compared a dual change model with freely estimated or unequal proportional change coefficients to a dual change model with equality constraints on these coefficients. The information in Table 2 is of two types. The upper portion provides fit indices for each model as a whole, and the lower portion provides parameter estimates and their associated critical ratios, the ratio of the parameter estimate to its standard error, similar to a t statistic. A critical ratio that approaches or exceeds an approximate absolute value of 2.00 is considered to be indicative of a salient parameter estimate; this corresponds to a ≥ 1.96 evaluation of a null hypothesis test with $p < .05$. The results for PTSD in Table 2 suggest that the parameter estimates in both variations of the dual change model, can be considered salient, with the exception of the covariance between initial status and constant change (where the critical ratios fell below 2.00). Of particular note for determining whether a dual change model is best (or whether one should consider the alternative proportional change model or constant change model) are the parameter estimates and critical ratios for the proportional change coefficients and the means and variances for the slope or constant change latent variable. Weakness in proportional change coefficients (again, as indicated by a low absolute value for the critical ratio) would suggest that prior status on the PTSD variable does not influence later status on that variable and would recommend dropping them and reverting to a possible constant change model.

[2] Any structural equation modeling program could be used. The specific Mplus scripts for the analyses reported here are available from the first author.

Table 1. Means, Standard Deviations, and Intercorrelations for Study Variables

Variable	*M*	*SD*	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
1. $PTSD_0$	23.73	13.23	–								
2. $PTSD_1$	21.66	13.81	.37	–							
3. $PTSD_2$	18.42	15.32	.22	.58	–						
4. $PTSD_3$	17.70	13.27	.14	.54	.71	–					
5. $STRAINS_0$	9.47	15.25	.06	−.10	−.33	−.44	–				
6. $STRAINS_1$	7.76	9.81	−.06	.01	.24	.02	.48	–			
7. $STRAINS_2$	10.93	12.04	.08	−.12	.14	−.20	.65	.62	–		
8. $STRAINS_3$	7.80	9.75	.05	.03	.18	−.12	.63	.73	.77	–	
9. AGE	12.89	3.70	−.16	.09	.20	.24	−.08	−.06	.03	−.04	—

Note. PTSD = posttraumatic stress disorder; STRAINS = family strains; 0–3 subscripts for PTSD and STRAINS indicate Time Classes 1–4, respectively. All bivariate correlation estimates are based on $N = 190$, and thus a value of $r > .14$ would have $p < .05$ and the expected value of its sampling distribution equals 0.

Weakness in the mean and variance of a slope would suggest no tendency for natural growth or change and would recommend removing the slope latent variable and reverting to a possible proportional change model. Weakness in both proportional change and slope would suggest no internal sources of change, that is, that prior status on the PTSD variable does not influence later status on that variable nor is there a tendency for natural growth or change.

Values of parameter estimates and corresponding critical ratios in Table 2 lead us to conclude that the dual change model is appropriate for the Saxe PTSD data. Moreover, the by-and-large stronger fit indices for the model with freely estimated (i.e., unequal) proportional change coefficients lead to the conclusion that it is the better of the two in characterizing the dual change process. Thus, using the dual change-unequal proportions information from Table 2, applying the parameter values to Equation 7, and

Table 2. Latent Difference Score Models for Posttraumatic Stress Disorder (PTSD)

Fit indices	PTSD: Dual change; unequal proportions		PTSD: Dual change; equal proportions	
χ^2/df	5.04/5		9.94/7	
CFI	.99		.94	
TLI	.99		.95	
RMSEA	.01		.05	
SRMR	.10		.13	
Parameter estimates	Est	CR	Est	CR
Initial status mean	23.48	21.81	23.71	22.10
Initial status variance	103.63	4.52	100.15	4.32
Constant change mean	15.31	4.46	11.91	3.12
Constant change variance	79.54	2.85	53.04	2.28
Initial status with constant change	29.47	1.66	22.26	1.41
Proportional change				
$PTSD_0$ –> LDS_{PTSD1}	−.71	−4.96	−.63	−3.80
$PTSD_1$ –> LDS_{PTSD2}	−.83	−4.89	−.63	−3.80
$PTSD_2$ –> LDS_{PTSD3}	−.87	−5.01	−.63	−3.80
Residual measurement error	74.12	6.57	79.56	6.63

Note. χ^2 = Chi square; *df* = degrees of freedom; CFI = comparative fit index; TLI = Tucker-Lewis index; RMSEA = root mean square error of approximation; SRMR = standardized root mean square residual; Est = estimated parameter; CR = critical ratio; $PTSD_0$ – $PTSD_2$ = latent PTSD scores; LDS_{PTSD1} – LDS_{PTSD3} = latent difference scores for PTSD.

adopting symbols that precisely define our PTSD outcome variable, we can write three regression equations, one for each of the three LDSs:

$$LDS_{PTSD1} = -.71\,PTSD_0 + 1.00s,$$
$$LDS_{PTSD2} = -.83\,PTSD_1 + 1.00s,$$

and

$$LDS_{PTSD3}, = -.87PTSD_2 + 1.00s, \quad (8)$$

where LDS_{PTSD1}, LDS_{PTSD2}, and LDS_{PTSD3} simply translate to the changes in PTSD for an individual from one time class to the next; $-.71PTSD_0$, $-.83PTSD_1$, and $-.87PTSD_2$ are the latent PTSD variables, each weighted by its unique proportional change coefficient; and s represents unit-weighted natural change or slope for an individual.

Results for family strains are given in the leftmost portion of Table 3. We again first fit a dual change model with unequal proportional change coefficients, followed by a dual change model with equal proportional change coefficients. In the former case, the value of the chi-square statistic was less than its degrees of freedom, resulting in both the comparative fit index (CFI) and the Tucker-Lewis index (TLI) approaching 1.00 and an RMSEA of .00, suggestive of perfect fit. All three proportional change coefficients were salient, as was the mean for constant change. Nonetheless, we attempted to simplify the model by constraining the proportional change coefficients to be equal. This latter model did not converge, likely due to its inability to accommodate the increase in family strains for Time Class 3. Similarly, we tried to fit a proportional change model with unequal proportional change coefficients and unequal LDS variances. This model was rejected because it yielded parameter estimates outside the range of acceptable values; two of three LDS variance estimates were negative. Thus, for family strains, we endorsed the original dual change model with unequal proportional change coefficients, shown in Table 3.

The next step in the process was to incorporate age as a time invariant predictor of change in PTSD. For our demonstration, this essentially involved simultaneously regressing each of the three LDSs for PTSD on the age variable, in the presence of prior status on PTSD and the constant change or slope variable. Alternative models for the influence of age could have been proposed based on theory, for example, that the effect of age is via an overall natural change trajectory, in which case the slope factor would be regressed on age. Returning to the current model, the influence of age was examined in the presence of the influences of autoregressive effects and nonstationarity, and the model for change simply added the influence of age at the time of the trauma:

$$LDS_{PTSD1} = -.61PTSD_0 + 1.00s + .44AGE$$
$$LDS_{PTSD2} = -.74PTSD_1 + 1.00s + .46AGE$$
$$LDS_{PTSD3} = -.99PTSD_2 + 1.00s + .85AGE \quad (9)$$

The elements are comparable to those in Equation 8, with the addition of age and its partial regression coefficients to characterize the unique contributions of each child's or adolescent's age to his or her changes in PTSD. Again, as revealed in the rightmost column of Table 3, the resulting model fit the data quite well. The effect of age was relevant in the prediction of the third LDS (critical ratio = 2.17), the shift in PTSD symptom severity from Time Class 3 to Time Class 4. It is noteworthy that the average slope for this model (6.83) is considerably smaller than the average slope in the selected model for PTSD without age (15.31). This suggests that much of the observed natural change in PTSD symptom severity can be accounted for by age of the child or adolescent at the time of the trauma.

Finally, we added the time-varying covariate of family strains to the model involving PTSD and age, to arrive at an integrated model that addressed our research question and included both time invariant (age) and time-varying (family strains) covariates in the prediction of changes in PTSD over time. The results are provided in Table 4. The chi-square statistic is lower than its degrees of freedom; the fit indices (CFI and TLI) approach 1.00, and the RMSEA takes on a value of .00. In this more complex model, there are a total of 31 estimated parameters: 9 for each of 2 dual change models for PTSD and family strains; 7 that

Table 3. Latent Difference Score Models for Family Strains and Posttraumatic Stress Disorder (PTSD) on Age

Fit indices	Family strains: Dual change; unequal proportions		PTSD on age	
χ^2/df	3.58/5		2.96/5	
CFI	1.00		1.00	
TLI	1.00		1.00	
RMSEA	.00		.00	
SRMR	.09		.04	
Parameter estimates	Est	CR	Est	CR
Initial status mean	9.93	6.98	23.72	22.02
Initial status variance	196.45	6.26	104.28	22.76
Constant change mean	10.89	3.27	6.83	1.21
Constant change variance	138.32	1.45	77.03	2.80
Initial status with constant change	141.49	2.27	26.73	1.47
Proportional change				
$STRAINS_0$ –> $LDS_{STRAINS1}$; $PTSD_0$ –> LDS_{PTSD1}	−1.31	−4.22	−.61	−3.62
$STRAINS_1$ –> $LDS_{STRAINS2}$; $PTSD_1$ –> LDS_{PTSD2}	−0.94	−2.11	−.74	−3.16
$STRAINS_2$ –> $LDS_{STRAINS3}$; $PTSD_2$ –> LDS_{PTSD3}	−1.25	−4.34	−.99	−5.63
Residual measurement error	28.29	5.10	72.43	6.47
Age-related effects				
Age mean			12.89	48.08
Age variance			13.66	9.75
Age with initial status			−7.62	−1.86
Age –> LDS_{PTSD1}			.44	1.31
Age –> LDS_{PTSD2}			.46	1.00
Age –> LDS_{PTSD3}			.85	2.17

Note. χ^2 = Chi square; df = degrees of freedom; CFI = comparative fit index; TLI = Tucker-Lewis index; RMSEA = root mean square error of approximation; SRMR = standardized root mean square residual; Est = estimated parameter; CR = critical ratio; $STRAINS_0$ − $STRAINS_2$ = latent family strains scores; $LDS_{STRAINS1}$ - $LDS_{STRAINS3}$ = latent difference scores for family strains; $PTSD_0$ − $PTSD_2$ = latent PTSD scores; LDS_{PTSD1} − LDS_{PTSD3} = latent difference scores for PTSD.

are age-related (mean and variance of age, covariance of age with initial status of PTSD and with initial status of family strains, and influences of age on the 3 LDSs for PTSD); and the 6 unidirectional associations that link PTSD and family strains (the 3 latent family strains variables predicting changes in PTSD and the 3 latent PTSD variables predicting changes in family strains). To address the research question that guided this demonstration, the following equations are pertinent:

$$LDS_{PTSD_1} = -.58PTSD_0 + 1.00s + .51AGE - .02\,STRAINS_0$$

$$LDS_{PTSD_2} = -.73PTSD_1 + 1.00s + .16AGE + .70\,STRAINS_1$$

$$LDS_{PTSD_3} = -.97PTSD_2 + 1.00s + .92AGE - .01\,STRAINS_2 \quad (10)$$

In Table 4 it can be seen that, as before, the three proportional change coefficients for PTSD (−.58, −.73, and −.97) have critical ratios that exceed the absolute value of 2.00. They may thus be considered important contributors to change in PTSD over each of the three time intervals. The constant change mean or average slope (5.46; critical ratio = 1.00) remains less important when age at time of exposure is included in the model, although there are individual differences in slope (variance = 77.88; critical ratio = 2.80).

Table 4. Integrated Latent Difference Score Model

Fit indices	Integrated model: PTSD, age, and family strains			
χ^2/df	21.25/23			
CFI	1.00			
TLI	1.00			
RMSEA	.00			
SRMR	.13			
	PTSD		Family strains	
Parameter estimates	Est	CR	Est	CR
Initial status mean	23.71	22.03	9.81	6.82
Initial status variance	109.47	22.60	198.25	6.26
Constant change (slope) mean	5.46	1.00	11.75	3.28
Constant change (slope) variance	77.88	2.80	161.34	1.42
Initial status with constant change	24.88	1.44	154.88	2.14
Proportional change				
$PTSD_0$–> LDS_{PTSD1}; $STRAINS_0$ –> $LDS_{STRAINS1}$	−0.58	−3.67	−1.37	−4.02
$PTSD_1$ –> LDS_{PTSD2}; $STRAINS_1$ –> $LDS_{STRAINS2}$	−0.73	−3.24	−1.14	−2.59
$PTSD_2$–> LDS_{PTSD3}; $STRAINS_2$ –> $LDS_{STRAINS3}$	−0.97	−5.36	−1.39	−3.89
Residual measurement error	66.75	6.25	27.30	5.11
Age-related effects				
Age with initial status	−7.67	−1.87	.13	.03
Age –> LDS_{PTSD1}	.51	1.50		
Age –> LDS_{PTSD2}	.16	0.33		
Age –> LDS_{PTSD3}	.92	2.28		
		Est	CR	
Age mean		12.89	48.08	
Age variance		13.66	9.75	
PTSD–family strains associations				
$STRAINS_0$–> LDS_{PTSD1}		−.02	−.08	
$STRAINS_1$–> LDS_{PTSD2}		.70	2.17	
$STRAINS_2$–> LDS_{PTSD3}		−.01	−.02	
$PTSD_0$–> $LDS_{STRAINS1}$		−.03	−.33	
$PTSD_1$–> $LDS_{STRAINS2}$		.05	.50	
$PTSD_2$–> $LDS_{STRAINS3}$		.06	.42	

Note. PTSD = posttraumatic stress disorder; χ^2 = chi square; *df* = degrees of freedom; CFI = comparative fit index; TLI = Tucker-Lewis index; RMSEA = root mean square error of approximation; SRMR = standardized root mean square residual; Est = estimated parameter; CR = critical ratio; $PTSD_0$ − $PTSD_2$ = latent PTSD scores; LDS_{PTSD1}- LDS_{PTSD3} = latent difference scores for PTSD; $STRAINS_0$ - $STRAINS_2$ = latent family strains scores; $LDS_{STRAINS1}$ - $LDS_{STRAINS3}$= latent difference scores for family strains.

The equation for the first latent difference score (LDS_{PTSD_1}), therefore, is interpreted as follows: Change in PTSD for any given individual is most strongly influenced by that individual's prior status on PTSD ($-.58PTSD_0$), such that higher scores at the prior assessment are associated with a greater reduction in symptoms. Regarding the influence of natural change ($1.00s$), the salient slope variance indicates important individual differences that should be included in predicting individual change. Controlling for these internal sources of change (autoregressive effects and natural growth), neither age ($.51AGE$) nor family strains ($-.02STRAINS_0$) appear to

impact change in PTSD, both having critical ratios that are below the conventional criterion (critical ratios = 1.50 and −.08, respectively).

However, for the second interval, scores on family strains were positively associated with a change in PTSD (coefficient = .70; critical ratio = 2.17), such that higher levels of family strains predicted a subsequent increase in PTSD symptom severity for the child or adolescent. And, for the third interval—the time period most remote from the traumatic event—an effect was observed for age (coefficient = .92; critical ratio = 2.28): Older children were less likely to exhibit a decline in symptom severity than younger children. These latter two influences are above and beyond, or "control for," autoregressive effects and nonstationarity, or natural change.

UTILITY OF THE LDS APPROACH FOR TRAUMA RESEARCH

What advantage does the LDS approach yield to the trauma researcher? Does the approach allow the investigator to discover relationships embedded in the data that might be obscured using other methods? The rather complex longitudinal Saxe data (used in the above demonstration) were generated to better understand how PTSD develops in children exposed to a traumatic burn or injury and the risk and resilience factors that impact symptom severity for this cohort of highly vulnerable children. The design called for PTSD assessment around the time of the trauma and then sequentially into the second year after the event. Initially, the emphasis was on the extent to which selected risk and resilience factors predicted the overall trajectory of PTSD symptom severity. In effect, such a growth curve methodology might obscure the possibility that a particular risk or resilience factor exerts a differential influence on change, depending upon the specific time interval or segment of the overall trajectory. In this regard, the LDS method may serve as a potent hypothesis-generating and hypothesis-testing tool for trauma researchers.

For example, in the sequence of LDS analyses using the Saxe data, we performed what essentially was a hierarchical multiple regression in which the three PTSD change scores were first simultaneously regressed on dual internal mechanisms of change (see Equation 8). Next, the fixed marker or time-invariant age at time of trauma variable was added (see Equation 9). Finally, the family strains covariate, measured repeatedly along with the assessments of PTSD, was included to examine the influence of this time-varying risk factor on change in PTSD symptom severity over various postexposure timeframes (see Equation 10). In this way, we were able to see there were differential influences of both age and family strains, depending upon the particular time interval being considered. Neither age nor family strains uniquely predicted change in PTSD symptom severity over the first interval of time following exposure. However, the family strains variable was noteworthy in the prediction of change in PTSD symptom severity from roughly 106 days to 196 days posttrauma, indexed to the medians of the time classes. Parent or guardian reports of family strains predicted an increase in the child's or adolescent's PTSD symptom severity during this interval. Furthermore, age at time of trauma was most important in predicting change in PTSD symptom severity from approximately 196 days to 376 days posttrauma, again indexed to the medians of the time classes. For older children, on average, there was less negative change or diminution in symptom severity during this interval.

It is, of course, beyond the scope of this article to provide a detailed interpretation of the Saxe LDS findings. Nevertheless, it was initially surprising that older children were more vulnerable to persistent PTSD in the post-196 day period. It is possible that the more plastic condition of the brains in younger children makes adaptation to their postinjury life easier than for older children. Hence, after a period, this increased ability to adapt may become expressed as a reduction in PTSD symptoms. It is also possible that hormonal changes related to puberty can lead to more persistent PTSD. Similarly, what might be the implications of the finding that family strains primarily exerted a deleterious effect on PTSD symptom severity during the 106- to 196-day period? As it happens, this timeframe corresponds to hospital discharge and family and community reintegration. Early on, during the first timeframe following the trauma, the hospital environment is significantly

more contained and structured than the environments to which injured children are typically discharged. Following discharge, children spend much more time with their families, and the family frequently experiences the stress of not having immediately available medical and nursing care. As time goes on, however, one would expect that families will adapt, and it makes sense that family strains in the final timeframe would be less influential. Such interpretations of the findings are, of course, speculative. Nevertheless, by disaggregating a growth curve, the LDS approach affords opportunity for such hypothesis generation and for more-refined studies to test these hypotheses.

SUMMARY AND CONCLUSIONS

This article has introduced the LDS approach to the analysis of longitudinal trauma data and demonstrated its use with data from a sample of children and adolescents who suffered burns or other injuries. To review, several important advantages of this approach make it well suited to examining the developmental process that characterizes trends in symptom severity among those exposed to extremely distressing life events. Primarily, unlike traditional change score analysis with simple difference scores, the LDS model affords an optimally reliable index of change by modeling change in perfectly reliable scores over a time series, hence reducing the likelihood of bias in the estimates of parameters that describe that change, and enhancing power. In addition, as noted earlier, it can be conceived as a disaggregation of a longer-term trajectory or growth curve into a sequence of latent difference segments, each of which is a potential outcome to be examined and understood. Thus, a single index of change or slope in a growth curve analysis becomes several to many change variables, each signifying change over a different interval in a times series. Consequently, more fine-tuned evaluations of the effects of putative risk and resilience factors—or theory-based components of an intervention—are available, in that one may appraise at what interval in a time series a particular factor is most pertinent. Moreover, by allowing for the estimation of internal sources of change (both autoregressive effects and natural change), an unambiguous interpretation of important external factors is possible. As pointed out previously, more traditional cross-lagged panel analyses take into consideration autoregressive effects, but not natural change.

The assumptions of this LDS approach are, of course, those of any structural equation modeling procedure. Endogenous variables must be continuous, and the residuals of all endogenous variables are assumed to be multivariate normally distributed. Violations of the normality assumption, such as would occur with ceiling or floor effects, or if endogenous variables were counts, or dichotomous or ordered polytomous data, would require nonstandard structural equation modeling procedures (see Muthén & Muthén, 2006). Furthermore, while the full information maximum likelihood estimator that is used will accommodate empty cells in the matrix of covariance coverage, there is always a limit on the degree of incomplete data. Too much "missingness" over time classes could result in the solution's failure to converge and/or a lack of identification of key parameters.

Although not elaborated here, the LDS approach can parsimoniously accommodate very complex patterns of responses over time. The illustration presented in this article allowed for variation in the proportional change coefficients but fixed the constant change coefficients at a value of 1. By freeing the constant change coefficients in conjunction with either freeing, fixing, or constraining proportional change coefficients, a variety of complex functions can be examined, for example, the up-and-down fluctuations in symptom severity among those who are chronically affected by posttraumatic conditions. Finally, the LDS approach may also be applied to treatment-outcome research to test hypotheses regarding differential influences of treatment over specific time intervals, either during the delivery of the intervention (e.g., sudden gains in symptom resolution) or over an extended timeframe postintervention (e.g., long-term maintenance of therapeutic effects).

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